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van Leeuwen, M.; van den Berg, S.M.; Hoekstra, R.A.; Boomsma, D.I.

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The Genetic and Environmental Structure of Verbal and Visuospatial Memory in Young Adults and Children

Marieke van Leeuwen
VU University Amsterdam, and The Netherlands Cancer Institute

Stéphanie M. van den Berg
VU University Amsterdam, and University of Twente

Rosa A. Hoekstra
VU University Amsterdam, and University of Cambridge

Dorret I. Boomsma
VU University Amsterdam

The extent to which verbal (VM) and visuospatial memory (VSM) tests measure the same or multiple constructs is unclear. Likewise the relationship between VM and VSM across development is not known. These questions are addressed using genetically informative data, studying two age cohorts (young adults and children) of twins and siblings. VM and VSM were measured in the working memory and short-term memory domain. Multivariate genetic analyses revealed that two highly correlated common genetic factors, one for VM and one for VSM, gave the best description of the covariance structure among the measures. Only in children, specific genetic factors were also present. This led to the following conclusions: In children, one genetic factor is responsible for linking VM and VSM. Specific genetic factors create differences between these two domains. During the course of development, the influence of genetic factors unique to each of these domains disappears and the genetic factor develops into two highly correlated factors, which are specific to VM and VSM respectively. At the environmental level, in both age cohorts, environmental factors create differences between these domains.

Keywords: verbal memory, visuospatial memory, development, cognition

The domain specificity versus the domain generality of working memory (WM) and short-term memory (STM) has been studied extensively in children as well as adults (e.g., Haavisto & Lehto, 2005; Maehara & Saito, 2007; Tillman, Nyberg, & Bohlin, in press). However, until now there is no agreement whether WM and STM are domain-specific or domain-general constructs. In other words, it is unclear whether there are domain-specific storage and executive function mechanisms for visuospatial and verbal memory tasks. We report on a twin study that examined the genetic and environmental relation between verbal and visuospatial memory in young adults and children from the general population. In both age groups, similar tasks were used to measure verbal and visuospatial WM and STM.

Marieke van Leeuwen, Stéphanie M. van den Berg, Rosa A. Hoekstra, Dorret I. Boomsma, Department of Biological Psychology, VU University, Amsterdam, The Netherlands. Marieke van Leeuwen is now at the Division of Psychosocial Research & Epidemiology, The Netherlands Cancer Institute, Amsterdam, The Netherlands. Stéphanie M. van den Berg is now at the Faculty of Behavioral Sciences, University of Twente, Enschede, The Netherlands. Rosa A. Hoekstra is now at the Autism Research Centre, Section of Developmental Psychiatry, University of Cambridge, Cambridge, United Kingdom.

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Correspondence concerning this article should be addressed to Marieke van Leeuwen, Postbus 90203, 1006 BE Amsterdam, The Netherlands. E-mail: M.v.leeuwen@nki.nl

Short-term memory is the capacity to store material over short periods of time in situations that do not impose other competing cognitive demands (Gathercole, Alloway, Willis, & Adams, 2006). Working memory is the system that is necessary for the concurrent storage and manipulation of information (Baddeley, 1992). Most studies (e.g., Kane et al., 2004) examine the domain generality versus the domain specificity of STM and WM from the perspective of the WM model of Baddeley (2000) and Baddeley & Hitch (1974). The Baddeley model comprises a *central executive* and three storage systems: the *phonological loop*, the *visuospatial sketchpad,* and the *episodic buffer*. The central executive is the system responsible for a range of regulatory functions, including attention, the control of action, and problem solving (Baddeley, 1996). The phonological loop comprises a phonological store that can hold memory traces for a few seconds before they fade, and an articulatory rehearsal process. The visuospatial sketchpad is its visuospatial counterpart (Baddeley, 2003). The episodic buffer provides temporary storage of information held in a multimodal code, which is capable of integrating information from a variety of sources, including long-term memory, into a unitary episodic representation. The buffer is episodic in the sense that it holds episodes whereby information is integrated across space and potentially extended across time (Baddeley, 2000). The episodic buffer is generally not included in studies on the domain generality versus the domain specificity of STM and WM.

Current consensus considers the phonological loop and the visuospatial sketchpad equivalent to STM (e.g., Alloway, Gathercole, & Pickering, 2006; Baddeley, 2003; Bayliss, Jarrold, Gunn, & Baddeley, 2003; Colom, Flores-Mendoza, Quiroga, & Privado, 2005; Conway, Cowan, Bunting, Therriault, & Minkoff, 2002; Gathercole, Pickering, Ambridge, & Wearing, 2004; Kane et al.,

2004). Unsworth and Engle (2007) concluded based on their literature review and meta-analysis that tasks measuring STM and WM largely measure the basic subcomponent processes, but differ in the extent to which these processes operate in a particular task.

The Domain Generality Versus the Domain Specificity of WM and STM

Haavisto and Lehto (2005) studied the domain specificity of WM in Air Force Recruits, by examining fluid/spatial and crystallized/verbal intelligence in relation to spatial and verbal WM. The findings showed that complex WM tasks measure separate domain-specific cognitive abilities, which supports the view that the executive functioning component of WM is domain-specific. After separating executive functioning from WM in children, Tillman et al. (in press) showed that visuospatial and verbal executive functioning both contribute independently to intelligence. This finding argues for a domain-specific view of WM in children. Thus, in children as well as in adults, there is evidence for domain specificity of WM.

These findings are in contrast with the Baddeley and Hitch model, which consists of one domain-general factor for executive functioning, and two domain-specific storage factors. Alloway et al. (2006), Gathercole et al. (2004), and Kane et al. (2004) demonstrated in their studies on the relation between WM and STM that domain-general executive function and domain-specific storage already exist at the age of 6 years, stay stable during development, and are also present in the adult general population. Maehara and Saito (2007) examined the domain specificity and domain invariance of WM in a student population, by looking at the effect of *stimulus order* and *processing time*. The authors compared the effect of processing tasks and storage in the same and different modalities. The results showed that next to domain-specific processes, domain-general processes also play a role in WM.

By demonstrating that disruption to STM does not depend on the modality of the interfering task, Jones, Farrand, Stuart, and Morris (1995) found support for the unitary model in adults. This model suggests one common factor for verbal and visuospatial STM (Jones, Beaman, & Macken, 1996). The study of Chuah and Maybery (1999) the study of Bayliss, Jarrold, Baddeley, Gunn, and Leigh (2005b) gave indirect evidence that the unitary model also applies to children.

Findings in studies on the domain generality and domain specificity of WM and STM are varying. These different views could be a result of studying different populations of participants. For example, Shah and Miyake (1996) and Kane et al. (2004) showed that the dissociation between spatial and verbal measures is less apparent in samples including individuals who are likely to vary widely in general ability (e.g., general population) than in samples including individuals with a restricted range of cognitive abilities (e.g., college students).

Hence, it is unclear whether these constructs are domain-general or domain-specific in nature. The contrasting findings are a reflection of the *molarity*-versus-*modularity* debate. In the molar view there is one system in which a unitary, general process functions across a wide variety of cognitive tasks. In the modular view there are numerous distinct cognitive processing units, each responsible for certain nonoverlapping cognitive tasks (Petrill, 1997). An explanation why cognitive studies find evidence for molarity as well as modularity is that cognition is influenced by genes and environment. There is evidence that genetic influences tie together diverse measures of cognitive functioning, whereas environmental effects drive wedges between different dimensions of cognitive processing (Luo, Petrill, & Thompson, 1994; Pedersen, Plomin, & McClearn, 1994). Genetic evidence points to molarity as evidenced by substantial genetic overlap across different cognitive abilities. In contrast, the different dimensions of cognitive functioning which consistently emerge across many studies seem to be primarily driven by environmental factors (Petrill, 1997). We hypothesize that this also applies to verbal and visuospatial WM and STM. Previous twin studies already showed that differences between individuals in performance on WM and STM tasks can be explained by differences in genotype (Blokland et al., 2008; Egan et al., 2001, 2003; Egan et al., 2004; Hoekstra, Bartels, Van Leeuwen, & Boomsma, in press; Kremen et al., 2007; Kuntsi et al., 2006; Polderman et al., 2006). Also, the genetic structure of verbal and visuospatial WM has been studied in young adults (Ando, Ono, & Wright, 2001). At the genetic level there was an indication for modality-specific and modality-invariant elements in WM.

Using multivariate genetic factor analysis we aim to establish to what extent the correlation between these constructs is caused by a common set of genes and/or environmental factors (Boomsma & Molenaar, 1986; Martin & Eaves, 1977), and whether the factor structure at the genetic level is consistent with the factor structure at the environmental level.

In two age cohorts, we address the question whether STM and WM are domain-general or domain-specific in nature. The older, young adult, cohort consists of 18-year-old twins coming from 186 families and the younger, child, cohort are 9-year-old twins and their siblings coming from 112 families. Verbal and visuospatial WM and STM are operationalized by administering one task in every domain. The genetic and environmental structure underlying the relation between verbal and visuospatial STM and WM is examined with multivariate genetic analyses (Boomsma & Molenaar, 1986; Martin & Eaves, 1977). By investigating their relationship in a genetically informative design, it is possible to elucidate the previous mixed findings, which, we hypothesize, are a result of genetic molarity and environmental modularity. Based on the existing literature, two models for the structure of verbal and visuospatial STM and WM are compared separately for the genetic and environmental factor structure:

1. STM and WM are overlapping, but modality-specific constructs. This will be reflected in a common factor for verbal memory and a common factor for visuospatial memory. The verbal memory and visuospatial memory factors will be correlated.
2. STM and WM are overlapping, modality-invariant constructs, and thus one common factor will describe the relation between visuospatial and verbal STM and WM.

By comparing model fit in the young adult and child cohorts we can get an indication whether there are any developmental changes in the underlying environmental and genetic structure of verbal and visuospatial WM and STM. We hypothesize that in young adults cognitive abilities are more differentiated and that this will be reflected at the genetic level only. This hypothesis ties in with the differentiation hypothesis, whose origins can be traced to the 'Law of Diminishing Returns' of Spearman (1927), and which

states that cognitive abilities become increasingly more differentiated during development (Garret, 1946; Reinert, 1970; Rietveld, Dolan, Van Baal, & Boomsma, 2003).

Materials and Method

Participants

Young adult cohort. Twin families were recruited via the Netherlands Twin Register (NTR, Boomsma et al., 2002, Boomsma et al., 2006). This cohort consisted of 186 families of 18-year-old twin pairs ($M = 18.2$, $SD = .21$) and one of their siblings ($N = 93$, $M = 18.5$, $SD = 5.74$) who take part in a longitudinal study of cognition and behavioral problems (Bartels, Rietveld, Van Baal, & Boomsma, 2002; Hoekstra, Bartels, & Boomsma, 2007). The group comprised 33 monozygotic male twin pairs (MZM), 34 dizygotic male twin pairs (DZM), 44 monozygotic female twin pairs (MZF), 38 dizygotic female twin pairs (DZF), and 37 dizygotic twin pairs of opposite sex (DOS). The zygosity of the same sex twin pairs was determined by DNA analyses (139 pairs), blood group polymorphisms (9 pairs), or longitudinally collected questionnaire items (Rietveld et al., 2000; 1 pair). There were 46 male and 47 female additional siblings in this cohort. The study was approved by the Central Committee on Research Involving Human Subjects (CCMO). When children were under 18, their parents signed an informed consent form. If they were aged 18 years or older, participants signed an informed consent themselves.

Data from one sibling were excluded from analyses, because this boy had severe learning difficulties. Data of the Corsi block tapping task of 33 participants (7% of the sample) were excluded because these participants had a score of 10 or lower. A score of 10 or lower means that they made mistakes when memorizing two or three blocks in a row, suggesting they probably did not understand the task properly.

Child cohort. The group of participants in this cohort consisted of 112 9-year-old twin pairs ($M = 9.1$, $SD = .10$) and one of their siblings age 9 to 14 years ($N = 100$, $M = 11.8$, $SD = 1.16$). Children were recruited from the NTR. This group takes part in a study on the development of cognition and brain structure (Van Leeuwen, Van den Berg, & Boomsma, 2008), and included 23 MZM pairs, 23 DZM pairs, 25 MZF, 21 DZF pairs, and 20 DOS pairs. For the same-sex twin pairs, zygosity determination was based on DNA polymorphisms (90 twin pairs), or on questionnaire items (2 pairs; Rietveld et al., 2000). There were 44 male and 56 female additional siblings. The study was approved by the CCMO, and parents signed an informed consent form for their children.

Three families did not complete the Corsi block tapping task, one sibling did not make the 2-back, and two siblings did not make the Wechsler Intelligence Scale for Children (WISC). Ten children were not able to complete the *n*-back task and eight children could not complete the Corsi.

Testing Procedures

In both cohorts all participants were individually tested at the VU University in separate rooms by experienced test administrators. For the young adult cohort, a testing day consisted of two parts; in the morning participants completed a medical test protocol, and after lunch they completed a psychological test protocol. The psychological test protocol including a break took about 3.5 hours to complete and included the Corsi block tapping task, the *n*-back task, and an intelligence test. Twins and siblings of 16 years of age and above completed the Wechsler Adult Intelligence Scale for Adults-III (WAIS-III; Wechsler, 1997), children under 16 were administered the Wechsler Intelligence Scale for Children-III (WISC–III; Wechsler et al., 2002). For the child cohort, a testing day consisted of a psychological test protocol only. Testing lasted for about 5 hours (including three breaks). Children completed as part of a larger test battery the Corsi block tapping task, the *n*-back task, and the WISC–III.

Corsi block tapping task. The Corsi block tapping task (Corsi, 1974) was included to assess short-term spatial memory. Participants sat in front of a touch screen monitor on which nine white blocks were displayed unevenly across a gray screen. In succession a number of blocks turned red for 1 s, after which the screen was blank for 3 s. After reappearance of the blocks, the participant had to tap the blocks on the screen in the same sequence in which they had changed color before. When a block was tapped, the block would turn red and stay that way until the end of the run. The computer registered each tap. Each participant was given two practice runs. In these practice runs each person had to memorize two blocks. Immediately after the practice runs the actual test was administered. Actual testing started with a series of two blocks. After every five runs, the item length was increased by one block. The test was terminated when the participant responded incorrectly to three out of five runs of the same length. The maximum number of blocks that could turn red in succession was nine. Performance was measured by total number of correct runs.

N-back task. Participants had to perform a spatial variant of the *n*-back task (Van Leeuwen, Van den Berg, Hoekstra, & Boomsma, 2007) to assess visuospatial WM. The *n*-back used in this protocol was designed after Gevins and Cutillo (1993) and Jansma, Ramsey, Coppola, and Kahn (2000), with increasing levels of difficulty. The participants were asked to look at an apple presented on a screen. The apple had four holes in which a caterpillar could appear. Participants were told to catch the caterpillar to prevent it from eating the apple, and were instructed to respond to the caterpillar by pushing one of four buttons with the thumb and index finger of both hands. The layout of the four buttons corresponded spatially to the four holes in which the caterpillar could appear. Participants had to indicate where the caterpillar was one move back (1-back), two moves back (2-back), three moves back (3-back), or four moves back (4-back). The caterpillar appeared in a hole for 1 s; after its disappearance there was a warning sound. Participants were instructed to respond after this warning sound and could respond until the next caterpillar appeared. Between two caterpillar moves, the apple was empty for 1 s.

Sessions were given in sessions of 20 trials. Each condition consisted of a practice session and three sessions in which performance was recorded. Practicing continued until the participants understood the task. After each session, participants received feedback on the number of apples they had saved from the caterpillar (correct responses) and how many had been eaten (incorrect responses). Following the feedback there was a break of 15 s. The task requires a continuous response to all stimuli and simultaneous monitoring and update of all movements of the caterpillar. Perfor-

mance on the task was scored by using the total number of correct responses. Maximum score per condition was 60.

In the young adult cohort, the 1-back and 2-back conditions were administered for practice purposes only, performance was recorded on the 3-back and 4-back conditions. For this cohort the sum score on the 3-back condition was used. Test–retest correlation of the 3-back condition in young adults is .70 (Van Leeuwen et al., 2007). In the child cohort, the 4-back condition was not administered and only the 1-back condition was used solitary for practice. Children were motivated during the task by counting the moves of the caterpillar. In the 2-back version, the test administrator counted continuously to three, and in the 3-back version, the administrator counted to four. For this cohort we used performance on the 2-back condition. For children the test–retest on 2-back is .65 (Van Leeuwen et al., 2007).

Digit span. Digit span forward (DSF) of the WAIS-III or WISC–III was used to measure verbal STM. In this task participants had to recall lists of numbers. The test started with a trial of two numbers. If participants recalled one out of two trials correctly, the list increased by one digit. Increments proceeded, until participants had both of two trials wrong. Performance was scored as the total number of correct trials. To measure verbal WM the digit span backward task (DSB) was used. This time the participants had to recall lists of numbers in reverse order. Test–restest correlation for digit span (forward and backward together) of the WAIS-III is .74 (Kooij, Rolfhus, Wilkins, Yang, & Zhu, 2004). The split half coefficient for the internal consistency of digit span of the WISC–III is .67 (Wechsler et al., 2002).

Data Analysis

All data analyses were performed using the software package Mx (Neale, Boker, Xie, & Maes, 2006). First, general covariance matrices, means, and the effect of sex and age on the means were estimated in a saturated model. Means were estimated separately for MZ twins, DZ twins, and siblings. The 12 × 12 covariance matrices (i.e., 4 variables × 3 family members) were estimated separately for MZ and DZ twin families. In the saturated model, separate covariances were estimated for MZ twin pairs, DZ twin pairs, and twin-sibling pairs. The phenotypic, MZ, DZ, and twin-sibling correlations were derived by standardizing the corresponding covariances. Since a large number of parameters were estimated, this model yields a good description of the data.

First, several assumptions, such as equality of means and variances in twins and siblings, were tested by fitting a series of nested models in which the means and variances for MZ and DZ twins and for twins and siblings were equated. The assumption that the four variables covaried in the same way within twins and siblings was tested by constraining the phenotypic covariances among measures to be the same in twins and siblings. Next, the assumption was tested that the resemblance in DZ twins is similar to the resemblance in nontwin siblings. We continued equating parameters until the most parsimonious model with still acceptable fit was established. The choice for the best fitting model was based on likelihood ratio tests. The difference between minus twice the log likelihoods ($-2\,LL$) of two nested models asymptotically follows a χ^2 distribution. The degrees of freedom are given by the difference in the number of parameters estimated in the two nested models. A high increase in χ^2 against a low gain of degrees of freedom denotes a worse fit of the submodel as compared with the full model. The means and the covariance structure between family members and between traits were tested for equality across the age cohorts. All data were analyzed, including data from families with incomplete twin pairs or without an additional sibling, using the raw data option in Mx.

Genetic modeling: Univariate analysis. To get a first impression of the relative influence of genes and environment on individual differences in memory performance, MZ, DZ, and sibling correlations were inspected. If MZ twin correlations are higher than DZ twin and twin-sibling correlations, part of the individual differences are caused by genetic effects, comprising *additive genetic effects* (A) and *nonadditive genetic effects* (D). If DZ twin and twin-sibling correlations are more than half the size of MZ correlations, the resemblance between twins is at least partly caused by *shared environmental effects* (C; environmental effects shared among offspring brought up in the same family). If MZ twin correlations are more than twice as high as DZ twin and twin-sibling correlations, D is likely to contribute to individual differences in memory performance. Differences within MZ twin pairs reflect the importance of *unique environment* (E). To have sufficient power to detect D or C large samples are required (Boomsma, Busjahn, & Peltonen, 2002; Plomin, DeFries, McClearn, & McGuffin, 2001). Based on the limited sample size and on inspection of the MZ, DZ, and twin-sibling correlations, we decided to fit a genetic model in which the relative contributions of A end E were estimated.

Formally, a trait or *phenotype* (P; i.e., observed characteristic of an individual that results from the combined effect of genes and environment) can be represented at the individual level as:

$$P_{ij} = a * A_{ij} + e * E_{ij},$$

where $i = 1, 2, \ldots 112$ (families) and $j = 1, 2$, or 3 (family members) and A and E are factors (latent variables, that are not observed directly). A and E are standardized to have unit variance. Figure 1 represents the phenotypes in one twin pair and one additional sibling in a genetic path model. $P_{Twin\ 1}$, $P_{Twin\ 2}$, and $P_{sibling}$ represent the phenotypes measured in these participants. The variance in P due to A and E is given by the square of a and e, respectively, so that $\mathrm{Var}(P) = a^2 + e^2$, which means that the observed variance in a population is attributed to variance caused by genes and variance caused by environment. Note that e^2 also contains variance due to measurement error.

MZ twins are practically identical at the DNA sequence level and therefore genetic effects are nearly perfectly correlated in MZ twins. DZ twins and siblings share on average half of their segregating genes so that the expected genetic correlation between their additive genetic effects (A) is 1/2. By definition, the correlation among the unique environmental effects (E) in twins and siblings is zero. Therefore the covariance within MZ twin pairs can be modeled as:

$$\mathrm{Cov}_{MZ}(P_{Twin\ 1}, P_{Twin\ 2}) = a^2,$$

and within DZ twin pairs and twin-sibling pairs as:

$$\mathrm{Cov}_{DZ}(P_{Twin\ 1}, P_{Twin\ 2}) = \mathrm{Cov}(P_{Twin\ 1}, P_{Sibling}) = 1/2\ a^2.$$

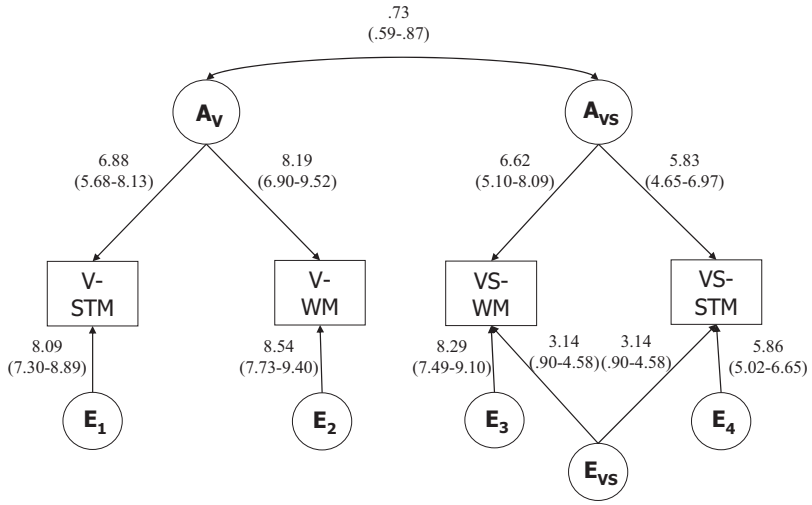

Figure 1. Best fitting genetic factor model in the young adult group with (unstandardized) factor loadings and their confidence intervals between brackets. A = genetic factor; E = environmental factor; V= verbal; VS = visuospacial; STM = short-term memory; WM = working memory.

Genetic modeling: Multivariate analysis. To determine to what extent the covariation among the four measures is due to correlated genetic and environmental effects, multivariate genetic factor analysis was applied. In a multivariate analysis, the *cross twin–cross trait* correlations between MZ and DZ twins and between twins and siblings contain information on the etiology of the association between traits. An example of a cross twin–cross trait correlation is the correlation between visuospatial WM in twin 1 and verbal WM in twin 2. These cross-correlations are estimated in the saturated model. Larger MZ cross-correlations compared to the DZ and twin-siblings cross-correlations indicate that part of the covariation between the two traits is determined by correlated genetic factors.

Additive genetic and unique environmental effects were modeled using a saturated four factor structure with all *factor loadings* (the loadings of the observed variables on the A or E factors) represented in two 4×4 lower triangular matrices (one for A and one for E). In a saturated factor structure, all possible contributions are parameterized; therefore it yields the best possible fit to the data. First, it was tested whether genes contributed significantly to the variation in and the covariation among the four measures. This was accomplished by assessing the deterioration of model fit of the saturated four factor model after the A factor was dropped from the model.

After establishing the significance of A, based on the existing literature, six models were evaluated, to asses the underlying A structure and E structure. Model fitting started with two models to asses the underlying A structure:

1. Two common genetic factors, one for verbal memory (VM) and one for visuospatial memory (VSM; VSM-VM model). The two genetic factors were allowed to correlate with each other.
2. One genetic factor model; one common genetic factor influences all four phenotypes. The assumption in this model is that all tasks are influenced by one set of genes.

In both models, the four variables could be influenced by genetic effects specific to that task. E was modeled as a lower triangular matrix. In this way, every possible contribution of E is modeled, and therefore all variance caused by E is captured. In Model 1 the correlation between the two common A factors was bound between zero and one. $\mathbf{A}_C$ is 4×2 matrix with the common genetic factor loadings, $\mathbf{A}_S$ is a 4×4 diagonal matrix containing the specific genetic factor loadings, and $\mathbf{E}$ is a 4×4 lower triangular matrix containing the unique environmental factor loadings. Within an individual, the variance in P (where P now is a four-variate phenotype) due to the two common A factors, the specific A factor and the saturated E structure is then given by:

$$\mathbf{V}_P = \mathbf{A}_C \times \mathbf{R} \times \mathbf{A}_C' + \mathbf{A}_S \times \mathbf{A}_S' + \mathbf{E} \times \mathbf{E}'$$

where $'$ indicates a transposed matrix, $\mathbf{V}_P$ is a 4×4 symmetrical variance/covariance matrix containing the variances of the four variables and the covariances between these variables, and $\mathbf{R}$ is a 2×2 standardized symmetrical matrix, with on the off-diagonal the correlation between the two A factors.

From the two models, the best fitting model was selected and subsequently model fit was improved by dropping parameters that did not significantly contribute to model fit. Consecutively, the same procedure was repeated for the factor structure of E: fitting the same two models for E with a saturated A structure. In the final model, the best fitting model for A was joined with the best fitting model for E.

Results

Means, standard deviations, and age and sex effects are reported in Table 1. Means were equal for MZ and DZ twins and siblings (young adult cohort: $\Delta\chi^2 = 7.850$, $\Delta df = 8$, $p = .45$; child cohort: $\Delta\chi^2 = 9.349$, $\Delta df = 8$, $p = .31$). There were no significant effects of sex on the means of the four tasks in the young adult or the child

Table 1
Maximum Likelihood Estimates of Means, SD and Age Regression of the Variables

Variable		*N*	Mean	*SD*	Age regression
		Young adult cohort			
V-STM	DSF	454	8.95	1.74	—
VS-STM	Corsi	421	19.34	3.51	.14
VS-WM	3-back	442	36.25	10.95	.20
V-WM	DSB	454	6.90	1.93	—

Variable		*N*	Mean	*SD* twins	*SD* sibs	Age regression
			Child cohort			
V-STM	DSF	322	7.23	1.53	2.15	.47
VS-STM	Corsi	310	12.80	3.91	4.78	1.24
VS-WM	2-back	313	29.69	10.40	15.77	3.38
V-WM	DSB	323	4.72	1.37	2.09	.45

Note. V = verbal; VS = visuospatial; STM = short-term memory; WM = working memory.

cohort. A significant effect of age on the means of the Corsi and *n*-back (performance increased with age) was found in the young adult cohort. In the child cohort, there was a significant age effect on the means of all variables. All subsequent models were corrected for these effects. Constraining the means, the within-person variance–covariance matrices and the between-person variance–covariance matrices across both cohorts resulted in significant deteriorations of fit (means: $\Delta\chi^2 = 298.750$, $\Delta df = 12$, $p = .00$; within person: $\Delta\chi^2 = 487.291$, $\Delta df = 20$, $p = .00$; between person: $\Delta\chi^2 = 56.892$, $\Delta df = 30$, $p = .00$). Therefore in all subsequent analyses data of the young adult and the child group could not be analyzed simultaneously.

In the young adult cohort, the variances and covariances among the four measures were equal for twins and siblings ($\Delta\chi^2 = 13.652$, $\Delta df = 10$, $p = .19$). DZ covariances and twin-sibling covariances could be equated ($\Delta\chi^2 = 8.494$, $\Delta df = 10$, $p = .58$). Therefore, in all subsequent genetic models DZ and twin-sibling covariances were equated. This way also twin-sibling covariance contributed to the estimation of A and E, which amplified the power of the study (Posthuma & Boomsma, 2000). In the child cohort, variances in twins and siblings could not be equated ($\Delta\chi^2 = 22.687$, $\Delta df = 10$, $p = .01$). In the siblings, there was more variation in the DSF, 2-back, and DSB. We corrected for this variance inequality by multiplying the variances of the DSF, 2-back, and DSB in the siblings by a factor that equated these three variances between twins and siblings. DZ covariation could be equated to twin–sibling covariation ($\Delta\chi^2 = 9.121$, $\Delta df = 10$, $p = .52$).

In both cohorts, the phenotypic correlations among variables were modest to moderate (see Table 2). In the young adult sample, phenotypic correlations were overall somewhat higher than in the child cohort. In the lower parts of Table 2 the MZ and DZ/twin-sibling correlations are displayed on the diagonal. On the left side of the diagonal, MZ correlations are reported, and on the right side, are the combined DZ correlations and twin/sibling correlations. MZ correlations were higher than DZ/twin-sibling correlations in both cohorts, indicating genetic influence on the variance of the four variables. Below the diagonal, MZ cross correlations, and above the diagonal, DZ/twin-sibling cross correlations, are presented. In both cohorts, most MZ cross correlations were higher than DZ/twin-sibling cross correlations, suggesting that genes play a role in the covariation among the four variables.

Model fitting results of the young adult cohort are presented in the top of Table 3. As was indicated by the higher MZ (cross) correlations than DZ/twin-sibling (cross) correlations, A could not be dropped from the four-variate AE model without a significant deterioration of fit ($\Delta\chi^2 = 57.426$, $\Delta df = 10$, $p < .01$). Therefore, it can be concluded that genes play a significant role in variation in, and the covariation among the four measures.

Next, the three four-variate factor models as described above were fitted for the A and E structure separately. Comparing the two models (VSM-VM model, and one common factor) for the underlying genetic structure revealed that the VSM-VM model was the best fitting model. In this model, two-genetic factor explained the genetic covariance among the four measures. All four specific genetic factors could be dropped from the model without a significant reduction of fit. Thus, none of the four measures was influenced by genes specific to that measure.

Fitting the two models on the underlying E structure revealed that the unique environmental influences were also best captured by the VSM-VM model. The verbal factor could be dropped without a significant deterioration of fit. Hence, only visuospatial WM and STM are influenced by the same environmental factor; this factor explains part of the covariance between these measures.

Thus, the final AE model in the young adult cohort consisted of two correlated factors for verbal and visuospatial memory explaining all genetic variance, one common environmental factor for the visuospatial memory tasks, and one specific E factor for each variable (see Figure 1 and Table 4). The factor loadings and their confidence intervals are given in Figure 1. The correlation between the genetic factor for verbal memory and visuospatial memory was .73. The environmental factor between the VSM tasks explained 20% of the observed correlation between the visuospatial tasks. All other phenotypic correlations and the remaining covariance between the VSM tasks were explained by the two common genetic factors. Approximately 36%–48% of the individual variation in all tasks could be explained by genetic variation. The remaining variation was explained by variation in unique environmental factors.

In the child cohort, dropping A from the four-variate AE model also led to a significant deterioration of fit ($\Delta\chi^2 = 373.661$,

Table 2
Phenotypic, MZ, and DZ/Twin Sibling Correlations

Variable		DSF	Corsi	n-back	DSB
		Young adult cohort			
Phenotypic correlations					
V-STM	DSF	1.00			
VS-STM	Corsi	0.27	1.00		
VS-WM	3-back	0.22	0.48	1.00	
V-WM	DSB	0.44	0.36	0.33	1.00
MZ and DZ/twin sibling correlations					
V-STM	DSF	.49/.17	0.03	0.03	0.09
VS-STM	Corsi	0.26	.38/.14	0.10	0.04
VS-WM	3-back	0.18	0.40	.31/.17	0.06
V-WM	DSB	0.41	0.39	0.25	.39/.08
		Child Cohort			
Phenotypic correlations					
V-STM	DSF	1.00			
VS-STM	Corsi	0.21	1.00		
VS-WM	2-back	0.17	0.31	1.00	
V-WM	DSB	0.26	0.31	0.22	1.00
MZ and DZ/twin sibling correlations					
V-STM	DSF	0.57/.20	.09	.10	.27
VS-STM	Corsi	0.13	0.58/.16	.10	.14
VS-WM	2-back	0.09	0.24	0.57/.16	.12
V-WM	DSB	0.30	0.36	0.24	0.40/.12

Note. Maximum likelihood estimates of phenotypic (upper parts) and MZ and DZ/twin-sibling correlations (lower parts) between the variables corrected for age and sex. On the diagonal on the left side the MZ correlations and on the right the DZ/twin-sibling correlations, below the diagonal MZ cross correlations and above the diagonal DZ/twin-sibling cross correlations. V = verbal; VS = visuospatial; STM = short-term memory; WM = working memory.

$\Delta df = 10$, $p < .01$). We first tested whether the genetic factor model obtained in young adults also gave a good description for the relation among visuospatial and verbal WM and STM tasks in children. Therefore we started model fitting with the final model of the young adult cohort (Model 8, Table 3 and Figure 1).

Model fitting results are presented in Table 3. The model that fitted best in the young adults led to a significant deterioration of fit compared to the saturated AE model. Adding specific genetic factors for each variable improved fitting results significantly. Further fitting showed that the common environmental factor for visuospatial memory, and the specific genetic factors for verbal WM and visuospatial STM could be dropped from the model, and that the genetic correlation between verbal and visuospatial could be fixed to 1. Consequently, the child model consisted of 1) one

Table 3
Model Fitting Results

Model	*df*	−2LL	cpm	$\Delta\chi^2$	Δdf	*p*	AIC
		Young adult cohort					
1. Four variate AE model	1745	12965.267					9475.267
2. 2 fac A (VSM-VM), spec A, sat E	1746	12967.279	1	2.012	1	.16	9475.279
3. Common fac A, spec A, sat E	1747	12974.632	1	9.365	2	.01	9480.632
4. 2 fac A (VSM-VM), sat E	1750	12971.277	2	3.998	3	.41	9471.277
5. 2 factor E (VSM-VM), spec E, sat A	1746	12965.221	1	−0.046	1	inc.	9473.221
6. Common fac E, spec E, sat A	1747	12966.064	1	0.797	2	.67	9472.064
7. Common fac for VS, spec E, sat A	1750	12970.547	6	5.326	4	.26	9470.547
8. 2 fac A (VSM-VM), one fac for E VS, spec E	1755	12981.129	1	15.862	10	.10	9471.129
		Child cohort					
1. Four variate AE model	1237	8777.668					6303.668
2. 2 fac A (VSM-VM), one fac for E VS, spec E	1247	8800.753	1	23.085	10	.01	6306.753
3. 2 fac A (VSM-VM), one fac for E VS, spec A, spec E	1243	8782.614	1	4.946	6	.55	6296.614
4. Common fac A, spec A for V-STM and VS-WM, spec E	1247	8791.585	4	8.971	4	.06	6297.585

Note. Best fitting model bold faced. −2LL = −2 log likelihood; *df* = degrees of freedom; cpm = compared to model; AIC = Akaike's Information Criterion; A = additive genetic factor; E = environmental factor; VM = verbal memory; VS(M) = visuospatial (memory); spec = specific, sat = saturated, fac = factor.

Table 4
Parameter Estimates of the Variance Due to the Additive Genetic (A) and Environmental Factors (E) in the Young Adult Cohort

	Young adult cohort			Child cohort		
	Unstandardized solution		Standardized solution	Unstandardized solution		Standardized solution
Variable	A	E	A (heritability)	A	E	A (heritability)
V-STM	47.32	65.48	.42	41.17	43.82	.48
VS-STM	33.97	44.15	.43	21.04	36.16	.25
VS-WM	43.79	78.59	.36	24.14	25.47	.28
V-WM	67.14	72.95	.48	20.59	48.27	.24

Note. Estimates are based on the best-fitting model. On the left of the table the unstandardized solutions and on the right standardized solutions. In the standardized solution A and E add up to 1.00. A = additive genetic factor; E = environmental factor; V = verbal; VS = visuospatial; STM = short-term memory; WM = working memory.

common genetic factor for verbal and visuospatial memory, which explained all genetic covariation among the four measures; 2) one specific genetic factor for verbal STM and one specific genetic factor for visuospatial WM, which explained additional genetic variance in these of the variables; and 3) a specific environmental factor for each variable (see Figure 2 and Table 4). Hence, all phenotypic correlation between WM and STM could be explained by the common genetic factor. The common genetic factors explained 35% of the genetic variance in verbal STM and 40% in visuospatial WM. Furthermore, 24%–48% of the variation in all tasks could be explained by genetic variance. Therefore, heritability estimates are lower in the child cohort than in the young adult cohort. The remaining variation in memory in children could be explained by variation in environmental factors unique to each variable.

Discussion

In this study we addressed the following two questions: Are verbal and visuospatial memory domain-general or domain-specific in nature? This question was investigated by looking at their relationship in a genetically informative design. Second, are there any developmental changes in the genetic and environmental factor structure of visuospatial and verbal memory? This question was addressed by studying the genetic and environmental structure of these constructs in young adults and children.

In both the young adult and child cohorts, low correlations were observed between the measures of verbal and visuospatial memory at the phenotypic level. In the young adult cohort, the intercorrelations were overall higher than in the child cohort. These correlations were comparable to the correlations observed in the studies of Alloway et al. (2006) and Gathercole et al. (2004). Since findings at the phenotypic level of this study are comparable to previous studies and there were no mean differences between twins and siblings, we think it is safe to assume that the findings in our twin sibling population are representative for the general population.

In the young adult cohort, about 40% of the individual variation in task performance could be explained by genetic factors. In the child cohort this was about 30%. Since STM and WM are related to IQ (Ackerman, Beier, & Boyle, 2005; Colom et al., 2005; Kane et al., 2004), this is in concordance with previous research showing that the heritability of IQ increases with age (Hoekstra et al., 2007). In the young adults, two different factor models were compared: a VSM-VM model and a one common factor model. These models were fitted to the genetic and the environmental structure of visuospatial and verbal memory. At the nonobserved

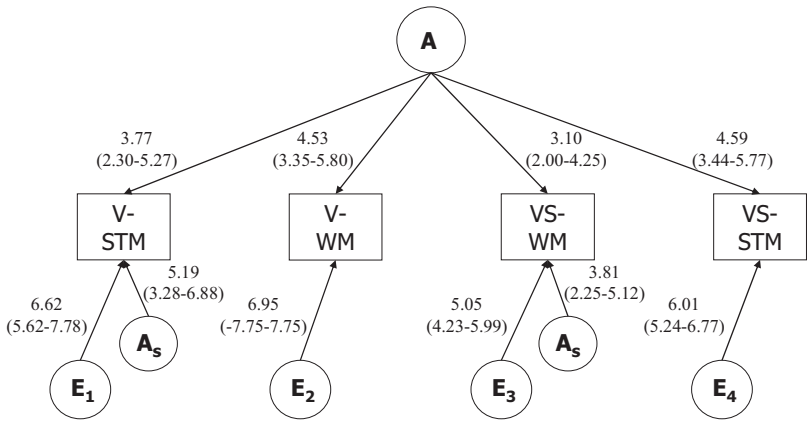

Figure 2. Best fitting genetic model in the child group with (undstandardized) factor loadings and their confidence intervals between brackets. A = genetic factor; E = environmental factor; V= verbal; VS = visuospacial; STM = short-term memory; WM = working memory.

(latent) level, two highly correlated common genetic factors were found, one for verbal and one for visuospatial memory, which explained most of the phenotypic correlations between visuospatial and verbal memory. A common environmental factor for visuospatial memory explained the remaining phenotypic covariance. All other environmental factors were uncorrelated. The specific environmental factors could reflect measurement errors unique to each task, or unique experiences that make people perform better on one task but not on the other. The common environmental factor can possibly be explained by the fact that both tasks were administered right after each other at the end of the testing day, and might therefore reflect weariness at the end of the day of testing in some participants. On the other hand, this common environmental factor can also be a true finding, since multiple studies report a higher correlation between visuospatial STM and WM, than between verbal STM and WM (Bayliss, Jarrold, Baddeley, & Gunn, 2005a; Miyake, Friedman, Rettinger, Shah, & Hegarty, 2001; Shah & Miyake, 1996).

In the young adult cohort, two genetic factors, one for visuospatial and one for verbal memory, explained most of the phenotypic correlations between the visuospatial and verbal memory tasks, indicating that these abilities are influenced by two related sets of genes. This suggests that at the genetic level, visuospatial memory and verbal memory are two different, but highly correlated systems. At the environmental level, variation was mainly explained by specific environmental factors, except for one common environmental factor for the visuospatial memory tasks. This means that variability in verbal and visuospatial STM and WM is caused by environmental influences specific to each of the variables. Environmental events (e.g., experience) make visuospatial and verbal WM and STM distinct cognitive processing units. This is in concordance with the neuroconstructivist view, which states that cognitive modules are a consequence of the developmental process of modularization and specific environmental interactions (Karmiloff-Smith, 2006, 1998).

Based on the fact that two highly correlated common genetic factors described the data best, it can be concluded that in young adulthood at the genetic level, separate storage and executive function mechanisms are at work for visuospatial and verbal information. (Haavisto & Lehto, 2005; Tillman et al., in press). The mixed findings reported in studies on domain specificity and domain generality of WM and STM could be a consequence of genetic molarity and environmental modularity. These findings are in concordance with the view of Price et al. (2000) and Petrill (1997), who suggest that genetic influences are responsible for linking diverse areas of cognitive functioning (genetic molarity), whereas environmental effects create differences between different domains of cognitive functioning (environmental modularity; e.g., Luo et al., 1994; Pedersen et al., 1994).

As in the adult cohort, in the child cohort, most of the phenotypic correlations were explained by genetics. Moreover, the study shows that in young adulthood, as well as in childhood, tasks measuring verbal and visuospatial memory are highly genetically correlated. However, the results in the children also indicated differences in the genetic structure of cognition in children as opposed to young adults: there is one common genetic factor for verbal and visuospatial memory; there are specific genetic factors for verbal STM and visuospatial WM; and there is no common environmental factor for visuospatial memory.

Although this study does not allow testing the differences between the child and the young adult cohort statistically, we will tentatively interpret the results that warrant interpretation. In children, it was shown that, apart from one common genetic factor, specific genetic factors also explain part of the variability in the two of the four abilities: each of these two abilities is also influenced by a genetic factor that does not influence the other abilities. This also explains why in the young adults, phenotypic correlations were overall higher than in the children. Based on this research one can only speculate what these specific genetic factors might be. One could think in the case of visuospatial WM of genes that influence the dorsolateral part of the prefrontal cortex, an area involved in visuospatial WM (Casey, Giedd, & Thomas, 2000). Thus in children, verbal and visuospatial memory are only partly overlapping abilities at this age. So, in contrast to our expectations, the correlation between genetic factors that represent different domains of cognition increases with age. A similar finding had been reported by Casto, DeFries, and Fulker (1995); Hoekstra et al. (2007); Price et al. (2000), and Rietveld, Dolan, Van Baal, and Boomsma (2003). They concluded that genetic effects on cognitive abilities may be more modular in early development and become increasingly molar later in life. This is in concordance with cross-sectional and longitudinal imaging studies of late childhood and adolescence, which show that brain regions with more basic functions mature first, followed by association areas involved in top down control. This developmental pattern is paralleled by a shift from a diffuse to more focal recruitment of cortical regions during cognitive tasks. This could be a consequence of learning and cognitive development (Casey, Tottenham, Liston, & Durston, 2005).

One limitation of this study is that only one measure for each construct was used. Because of that, test-specificity rather than construct-specificity could be responsible for the verbal and spatial memory factors. Using multiple indicators would have made our claims stronger. Also one could question whether we chose the best tasks to measure STM and WM. Maybe these tasks also measure verbal and visuospatial ability. However, in a longitudinal study design, it is not feasible to let children return multiple times to finish one test battery without a significant loss of participants on future test occasions. Another limitation of this study is that, as a consequence of distributional differences between the samples, we had to analyze data of these samples separately. However, to detect subtle differences in heritability between two cohorts, very large sample sizes are needed (Martin, Boomsma, & Machin, 1997), and the sample size of the present study would have been insufficient to do so. This could have weakened our claim regarding the developmental trajectory of the verbal visuospatial relation. In the future, large longitudinal studies are needed to further investigate the developmental trajectory of verbal and visuospatial memory. Nevertheless, the current study did have enough power to show that in contrast to the young adults, specific genetic factors in children did contribute significantly to the variation in the memory measures.

Based on this study, we can speculate what the common genetic factors for visuospatial and verbal memory represent. From twin studies it is known that brain structure is highly heritable (Baaré et al., 2001; Hulshoff Pol et al., 2006): differences in brain structure between people are caused by genetic variability between people. The study of Posthuma et al. (2003) showed that WM performance and brain volumes are genetically related. Since there is a genetic

relation between memory performance and brain structure, it is possible that the genetic correlation between verbal and visuospatial WM and STM represents processing of STM and WM by the same brain structures. The two different factors for verbal and visuospatial memory probably reflect processing by, respectively, the visual and auditory cortex. Whether this truly is the case should be addressed by future studies combining measures of brain structure and memory performance. For future studies, it would also be of interest to learn more about what kind of processes are captured by the two common genetic factors. Do they capture storage, executive functioning, general intelligence, or a combination? Another direction of research would be to take a closer look at the specific genetic factors found in children: what do they represent and when do they disappear? Also it is important to see if these findings can be replicated using different STM and WM tasks. Finally, by following the child group longitudinally we can establish if the effect of increasing genetic correlations between memory measures with age can be replicated.

To conclude, two major findings were obtained in this study. First, in young adults, two genetic factors are responsible for explaining the association between verbal and visuospatial WM and STM, whereas environmental factors create differences between these domains. This means that performance on visuospatial and verbal WM and STM is influenced by two highly correlated sets of genes. Therefore, from a genetic viewpoint one could say that verbal and visuospatial information are processed using two partly overlapping memory pathways. Second, the pattern of results between the two samples suggest that domain-specific genetic effects may be more prominent in childhood than in young adulthood, but that conclusive evidence of disappearing domain-specific genetic effects awaits confirmation in a longitudinal design.

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